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EXTRA-ALVEOLAR AIR RESULTING FROM SUBMARINE ESCAPE
TRAINING: A POST-TRAINING ROENTGENOGRAPHIC
SURVEY OF 170 SUBMARINERS

by

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EXTRA-ALVEOLAR AIR RESULTING FROM SUBMARINE ESCAPE TRAINING: A Post-Training Roentgenographic Survey of 170 Submariners

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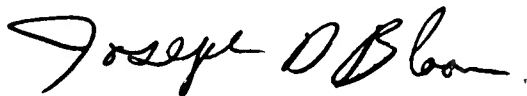Research Work Unit MF022.03.03-9025.32

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SUMMARY PAGE

THE PROBLEM

To investigate the incidence of asymptomatic extra-alveolar air in submarine enlisted men immediately after their completion of routine submarine escape training.

FINDINGS

The method of production and avenues of dissection of extra-alveolar air are described. The importance of the mediastinum as an area from which extra-alveolar air may dissect in several directions is then discussed. After critical review and analysis of a previous study and review of submarine escape training casualties, it is postulated that air emboli occur in approximately 72 percent of all casualties in whom extra-alveolar air has been demonstrated or is thought to exist. When a significant incidence of asymptomatic extra-alveolar air is found in personnel immediately after their completion of uneventful submarine escape training, it is postulated that the incidence of air emboli in this group is significantly higher than has been suspected.

APPLICATIONS

The results of this study should demonstrate that the potential hazards associated with submarine escape training are not insignificant, and that air emboli and extra-alveolar air resulting from such training probably occur with a far greater frequency than has been reported in earlier studies.

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ABSTRACT

Submarine escape training for submarine personnel has been conducted by the U. S. Navy since 1930. By 1957, more than 250,000 ascents had been recorded. Most casualties due to this training involved extra-alveolar air and/or embolism, and those few casualties which have occurred had usually been recorded and reported. In view of this, several questions were asked, among them one concerning the incidence of extra-alveolar air after submarine escape training in personnel who are not considered to be casualties.

Chest roentgenograms of 170 personnel taken immediately after undergoing routine submarine escape training were examined. Two men were found to have roentgenographic evidence of extra-alveolar air. The close association of extra-alveolar air with air embolism is postulated and the true incidence of these entities after submarine escape training is questioned. It was concluded that the incidence of air emboli and/or extra-alveolar air following submarine escape training may be much greater than previously suspected.

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INTRODUCTION

Bouyant ascent and Steinke Hood ascent training for submarine personnel in the U. S. Navy has been conducted at the Naval Submarine Base New London, Groton, Connecticut, and the Naval Submarine Base Pearl Harbor, Hawaii, since 1930 and 1932, respectively. The procedures used are well known and have been well studied. The personnel instructing and supervising ascents made by untrained submariners are thoroughly experienced in the techniques used during submarine escape training and are also well versed in the respiratory anatomy and diving physiology pertinent to such training. A minimal number of severe or fatal accidents related to submarine escape training have occurred. Those casualties which have resulted from other than physical trauma are due to the presence of air outside the normal respiratory pathways.

Numerous studies are available describing aviators bends and its pathogenesis, incidence, morbidity, and management. However, few studies have been published on the less spectacular subject of extra-alveolar air as manifest by pneumothorax, pneumomediastinum, pneumopericardium, and subcutaneous emphysema. In this study, an attempt was made to detect any evidence of asymptomatic extra-alveolar air in submarine personnel immediately after their completion of submarine escape training, by way of posterior-anterior and left lateral chest roentgenograms.

THE PROBLEMS

This study was prompted by the author's discussions with several submarine escape training instructors and physicians; all of whom described personal instances of chest and/or neck crepitation after several hours of diving duty at the Naval Submarine Base New London in the Submarine Escape Training Tank. The author also had occasion to examine two patients in the Naval Sub-

marine Medical Center Emergency Room with documented pneumomediastinum and subcutaneous emphysema of the chest and neck. Both patients had undergone uneventful bouyant ascent and Steinke Hood ascent training in the 24-hour period immediately prior to the onset of their symptoms. In view of the above circumstances, the following questions were asked:

(1) Does a detectable quantity of extra-alveolar air exist in personnel immediately following uneventful submarine escape training?

(2) Is extra-alveolar air, as manifest by subclinical or asymptomatic pneumothorax, pneumomediastinum, pneumopericardium, or subcutaneous emphysema, more common after routine submarine escape training than current knowledge would indicate?

(3) Are pre- and post-ascent chest roentgenograms feasible as a screening procedure for detection of extra-alvelor air resulting from submarine escape training?

(4) Is there any correlation between the presence of extra-alveolar air and air emboli in personnel following routine submarine escape training?

A discussion of basic anatomy begins the search for the answers to these questions.

DISCUSSION: ANATOMICAL AND HISTORICAL

The early development of both pleural and pericardial cavities begins with the invagination of three underinflated serous sacs (one for the pericardial and two for the pleural cavities), each of which is located in a bed of connective tissue known as the mediastinal matrix. If one indents each of these sacs with a finger, he invaginates the sac until both sides of the sac are approximated. Thus, a visceral layer is formed adjacent to the finger, and a parietal layer is formed and

represented by the opposite side of the visceral layer. The reflection of these two layers occurs at the base of the invaginating finger. The cavities formed (two pleural and one pericardial) are thus the space, potential or real, located between the visceral and parietal layers.[1,2]

For the pleural sacs, the invaginating finger represents the bronchi, pulmonary vessels, and lungs. For the pericardial sac, the invaginating finger represents the aorta and pulmonary vessels merging into the heart. The fibrous pericardium later develops from connective tissue external to the parietal layer of the pericardium. The potential connection of each of these three sacs is the mediastinal matrix. This matrix subsequently becomes the mediastinum in the mature state, and thus represents a potential link between the pericardial cavity and the two pleural cavities. In addition, the connective tissue of the neck, mediastinum, and retroperitoneum forms one vast continuum that includes the aorta and esophagus posteriorly. Thus, any air originating in one area of the continuum is, theoretically, free to dissect to any other area of the continuum.

In the mature state, the pleural and pericardial cavities are in essential contact, but are still separated by a plane of mediastinal connective tissue that includes; the phrenic nerves, the pericardiocophrenic vessels, and the epipericardial fat pads. This plane represents an avenue for dissecting air to follow and is visualized roentgenographically in a patient with pneumomediastinum as a fine radiolucent stripe immediately adjacent to the heart. An identical radiolucent stripe appears when the air is not located in the mediastinum, but is instead located within either pleural cavity, as with a pneumothorax.

A fine hair-line of soft tissue density is visualized roentgenographically just lateral to the radiolucent stripe of both above conditions. In a paracardiac pneumothorax, this density is composed entirely of visceral pleura, while in pneumomediastinum, both visceral and parietal pleura are seen. Differentiation between these pleural surfaces is not roentgenographically possible.[2]

When mediastinal air originates from the lung, its escape can be traced to the basic anatomic structure of this organ, the alveolus. Two types of alveoli exist: (1) Marginal alveoli, which lie adjacent to the perivascular sheaths and have no safety valves or intercommunicating pores. (2) Partitional alveoli, which lie adjacent to other alveoli and have safety valves and intercommunicating pores. Over-distention and subsequent rupture of partitional alveoli merely introduces air into other alveoli; however, over-distention of the marginal alveoli allows intra-alveolar pressure to become greater than that within the adjacent vessels. The pressure gradient may then result in alveolar rupture, with escape of air into the perivascular sheaths producing pulmonary interstitial emphysema.[3]

The theory of "pulmonary alveoli over-distention" as postulated by the Macklins best explains what has been found clinically and in animal experiments. In his 1939 studies, C. C. Macklin injected air under pressure into the trachea of anesthetized laboratory animals. Subsequent examination of the lungs of these animals revealed air between the outer walls of the arterioles and veins and their outer connective tissue boundaries.[4,5] The Macklins observations were confirmed in 1952 by Tocker and Langston[6]

Air under pressure in a perivascular sheath has been shown to dissect proximally toward the hilum and mediastinum, distally toward the visceral pleura, or in both directions.[7] In addition, when air escapes from the alveoli due to a significant differential between the intrapulmonic and intraarterial pressures, the air may rupture smaller pulmonary vessels and enter the blood stream thus producing air emboli.[8]

Thus pulmonary interstitial emphysema can lead directly to air embolism, pneumomediastinum, and pneumothorax, and indirectly (via the mediastinum) to subcutaneous emphysema of the neck, chest, and retroperitoneum as well as pneumothorax. But what about pneumopericardium? Does this ever occur secondary to pneumomediastinum?

Several cases of documented pneumopericardium, with pneumomediastinum as the presumed cause, have been reported in in-

fants. However, no cases have been reported in older children or adults.[9] The potential exists, anatomically, for the passage of air from the pulmonary interstitium to the mediastinum and thence to the pericardial cavity, either through visceral pericardial (sub-epicardial) dissection or through subparietal pericardium with resulting perforation into the pericardial cavity.[10] In Macklin's original experiments, however, air from the pulmonary alveoli traveling to the mediastinum never reached the pericardial cavity.[4]

A possible explanation for the above findings is the presence of an unusual susceptibility to dissection and rupture in the layers of serosal pericardium of infants. Cimmino resolves the question somewhat by stating that pneumopericardium associated with closed injury or with air infiltrating from the mediastinum should be diagnosed only with full consideration of the thickness of the normal parieto-fibrous pericardium with its associated soft tissues, and of the height of its reflection on the aorta and pulmonary artery.[1]

Thus, except for pulmonary interstitial emphysema resulting in pneumothorax of the ipsilateral pleural cavity, air embolism, and possibly pneumopericardium, all of the conditions under discussion rely upon the mediastinum as a pathway for the air involved. Four routes by which air may enter the mediastinum have been described: (1) the fascial planes of the neck, (2) tracheal, esophageal, or bronchial perforations, (3) the retroperitoneal space, and (4) pulmonary interstitial tissues. The latter appears to be the most common route.[11] In addition, Lam states that while mediastinal air may rupture into the pleural cavities, air in the pleural cavities never ruptures into the mediastinum.[12]

In summary, extra-alveolar air, once formed in the pulmonary interstitial tissue, has the capability of traveling in three directions: (1) intravascularly, (2) toward the pleural cavity of the affected side, and (3) toward the mediastinum. Intravascular migration of air results in air emboli. Air migrating toward the pleural cavity will produce a pneumothorax, and on occasion may mimic mediastinal air on a posterior-anterior (PA) roentgenogram of the chest. Air traveling along the perivascular sheaths to the mediastinum will produce pneumomediastinum. However, once extra-alveolar air reaches the mediastinum, it is capable of dissecting or rupturing its way into the pericardial cavity, either pleural cavity, around the aorta and/or esophagus into the retroperitoneal connective tissue, or into the subcutaneous connective tissues of the face, neck and/or thorax. Thus, detectable free air in the mediastinum becomes significant as a possible indication of similar extra-alveolar air elsewhere, and if extra-alveolar air is known to exist, air emboli could also be present. The consequences of this could obviously be far more serious.

DISCUSSION: RELATED STUDIES

There are a paucity of available studies which pertain to production of extra-alveolar air due to air expansion secondary to changes in ambient pressure. Altitude decompression studies by Stonehill, et al, have shown that pulmonary bullae expand with decreasing pressure.[13] In studying one patient with a benign air cyst without demonstrable bronchial communication, Parker, et al, demonstrated roentgenographically that the cyst expanded with increasing altitude and regressed with descent.[14] Tomashefski, et al, worked with several patients with pulmonary blebs and bullae. He observed that in ascents up to altitudes of 18,000 feet at the rate of 1,000 feet per minute, no distention or rupture of the blebs or bullae were noted on chest roentgenograms. He also concluded that spontaneous pneumothorax at altitude should occur no more frequently than at ground level. This conclusion was based on the fact that extensive communications occur between blebs, bullae, and emphysematons areas and may explain why over-distention and rupture of such lesions does not occur.[15]

Rapid compression and decompression of air occurs readily in diving and other underwater activities. Reynders described a 27 year old male who developed roentgenographically documented interstitial, mediastinal, and subcutaneous emphysema following repeated free dives and Self-Contained Underwater Breathing Apparatus (SCUBA)

dives to a depth of ten feet. Both the total number of dives and the total time spent in the water was not reported. Sharp right subscapular and anterior neck pain, substernal pain, and pain with swallowing all occurred while the subject was still in the water and subsequently disappeared over the following 36 hour period. Reynders concluded that more such accidents can be expected due to the increasing popularity of underwater sports.[16]

A study involving a review of all recorded cases of casualties in individual submarine escape training has been done by Moses.[17] Sixty-two cases of casualties resulting from three types of ascent training are noted with a summary of each case recorded in clinical reports or on form NAVMED 816, the U. S. Navy's Diving Accident Report. Moses' study covers the period from 1928 to 1957 and includes cases from the Escape Tank at Submarine Base, New London (39), the Escape Training Tank, Pearl Harbor (11), the Mine Tank at the Naval Gun Factory (3), Naval Medical Research Institute (1), and the open sea (8). Ascents were made from depths ranging from 18 to 110 feet. Buoyant ascent, free ascent, and ascents using a submarine escape apparatus (S.E.A. or "Momsen Lung" —essentially a closed-circuit SCUBA with a reservoir bag, a flutter valve for venting expanding gas in the bag on ascent, and a canister with carbon dioxide absorbent) were all associated with casualties.

In Moses' casualty group, 22 patients had the signs and/or symptoms of extra-alveolar air, present as pneumomediastinum and subcutaneous emphysema. Pneumothorax was also reported in two of these 22 patients. Associated air embolism occurred in 16 of the 22 cases (73 percent), with the times of onset of signs and symptoms noted no later than ten minutes after surfacing in each case. One non-air embolism casualty was noted to have X-ray evidence of pulmonary histoplasmosis, but no referral is made to intrathoracic calcification in any of the remaining 21 casualties. Also, no references were made concerning the presence or lack of chest roentgenograms in this group. Any one of the following was considered to be diagnostic of pneumomediastinum even though clinical examination or chest roentgenograms did not reveal the air: (1) substernal pain, (2) substernal fullness, (3) chest pain, or (4) chest fullness. In only three cases could extra-alveolar air be demonstrated roentgenographically. Moses states, "In a review of pre- and post-ascent chest X-ray (sic) on 100 buoyant ascents, British physicians noted subclinical mediastinal emphysema in one of the first 70 cases. The actual incidence of such an occurrence is only speculation. . ."

A more critical analysis of Moses' statistical data concerning escape training experience at the New London Tank has been made by this author by utilizing the clinical summaries of the 30 casualties reported at New London between August, 1930 and April, 1957. The statistics from the New London submarine escape training tank are thought to be more significant than are Moses' overall statistics because Submarine Base New London has always maintained the complete and careful records concerning the total number and type of ascents as well as the clinical summary of each casualty. It also is the oldest and most used tank for submarine escape training and trains both new submarine recruits as well as experienced submariners.

Moses' statistics from the New London Tank, using only casualties relating to the three methods of individual submarine escape, are shown in Table I for casualties involving air embolism only, air embolism in association with extra-alveolar air, and extra-alveolar air only. Twelve of the 30 casualties recorded had findings of air embolism only, with no reported evidence of extra-alveolar air. However, of the 18 casualties recorded with extra-alveolar air, 13 casualties (72 percent) occurred in association with air embolism. Table II, compiled from New London records, shows those 13 casualties in relation to the three methods of individual submarine escape used in training.

It is important to note that the diagnosis of extra-alveolar air in each of these 13 casualties was made without a chest roentgenogram. In five casualties, the diagnosis was established by finding subcutaneous emphysema on physical examination. However, in

ASCENT TYPE	CASUALTIES PER TOTAL ASCENTS	FATALITIES PER TOTAL ASCENTS	FATALITIES PER TOTAL CASUALTIES
S.E.A.	13/193,000=.007%	1/193,000=.001%	1/13=7.69%
Free	15/16,500=.090%	2/16,500=.012%	2/15=13.3%
Bouyant	2/6,500 =.030%	1/6,500 =.015%	1/2 =50%
Total (All Types)	30/216,000=.014%	4/216,000=.002%	4/30=13.4%

the remaining eight casualties, either substernal pain or fullness, or chest pain or fullness were considered to be direct evidence of pneumomediastinum and thus the presence of extra-alveolar air. The consistency of this practice over a 27-year period hopefully would indicate that this presumptive diagnosis is reasonably accurate. This in turn would indicate that air emboli, regardless of their size or extent, occur in approximately 72 percent of submarine escape training casualties with extra-alveolar air.

ASCENT TYPE	CASUALTIES PER TOTAL ASCENTS	FATALITIES PER TOTAL ASCENTS	FATALITIES PER TOTAL CASUALTIES
S.E.A.	4/193,000=.002%	1/193,000=.001%	1/4 = 25.0%
Free	8/16,500=.050%	1/16,500 =.006%	1/8 = 12.5%
Bouyant	1/6,500 =.015%	0/6,500 = NONE	0/1 = NONE
Total (All Types)	13/216,000=.006%	2/216,000=.001%	2/13=15.4%

With the absence of roentgenographic evidence of free air in casualties nonetheless diagnosed as having extra-alveolar air, one then questions whether or not errors of omission were committed in the 12 casualties where air embolism was considered to have occurred alone, without associated extra-alveolar air. In this group of 12 casualties, all but two presented with either convulsions, loss of consciousness, or paralysis as the initial manifestation of air embolism. Two cases were rapidly fatal. Central nervous system symptoms were noted in all 12 casualties. It is possible that in the rush to provide treatment for these more severe casualties, a relatively minor finding such as subcutaneous emphysema or a mediastinal crunch could

have been overlooked or gone undetected. Indeed, if rapid recompression were undertaken for treatment, as was done in most cases, the obvious signs of extra-alveolar air would rapidly disappear and the diagnosis would probably not be made. Subjective diagnostic criteria for extra-alveolar air, as earlier describe, would surely seem minor to a patient following unconsciousness or a Grand Mal seizure, if indeed such criteria were experienced. In summary, perhaps extra-alveolar air can be considered to occur in 72 percent of those casualties with air emboli.

Thus, for both groups of casualties discussed, the simultaneous presence of extra-alveolar air and air emboli would occur in 72 percent of the total casualties reported at New London. If air embolism and extra-alveolar air can exist to that extent in casualties due to submarine escape training, then perhaps the incidence of extra-alveolar air in non-casualties would reflect more accurately the true incidence of air embolism in such training. The incidence of asymptomatic extra-alveolar air due to submarine escape training has recently been determined by this author and is the subject of this report.

PROCEDURE

Between March 1967 and February 1968, chest roentgenograms were taken of 170 Basic Enlisted Submarine School Students between the ages of 17 and 24 years who underwent and successfully completed submarine escape training at New London, Connecticut. All students claimed to have made uneventful ascents and all students denied any unusual symptoms upon completion of their training. All roentgenograms were taken no later than one hour after completion of escape training.

The overall health of each student was excellent. Each student had been subjected to pressures of 50 pounds per square inch above atmospheric pressure (equivalent to a water depth of 112 feet) in a dry compression chamber test, and all had passed this test without incident. Each student had also been designated as physically qualified for submarine duty. Each student had previously been

screened by a physician, and any student having any acute chronic disease which could complicate escape training or possibly produce extra-alveolar air or air emboli was not allowed to undergo submarine escape training.

All training occurred in mid-morning or early afternoon and lasted approximately two hours. After a thorough indoctrination and instruction session, each student made two buoyant ascents followed by one Steinke Hood ascent, all from a depth of 50 feet. The average ascent rate for both types of ascents is known to approximate 5.5 feet per second. Duration of ascent for both types of ascents is 8-10 seconds. This value range represents the time interval between leaving the 50-foot depth and arriving at the surface.[18,19]

Two different types of patient positioning for roentgenographic study were used in an attempt to compare the effectiveness of both types as a routine screening procedure for detection of extra-alveolar air. PA chest roentgenograms were taken of 72 students, while both PA and left lateral chest roentgenograms were taken of the remaining 98 students. All films were then carefully reviewed by the author with special attention given to the mediastinum, cardiac borders, pulmonary apices, and soft tissues of the neck and thorax. Any evidence of extra-alveolar air, intrathoracic calcification, or scarring were noted. Recent pre-ascent training PA chest roentgenograms on each student were readily available for study and comparison. Subsequent comparisons of pre- versus post-ascent films were made for 38 students with questionable findings on the latter films. All questionable films were then studied and reviewed with Richard Dunbar, M.D., a Board-certified radiologist and Chief of Radiology at the Naval Submarine Medical Center. The decision was then made, entirely on a roentgenographic basis, regarding the presence or absence of extra-alveolar air in each student. Standard roentgenographic criteria for extra-alveolar air, as noted by Paul, Meschan, and Cimmino, were used for any diagnosis of extra-alveolar air made in this study.[1,20,21]

RESULTS

Table III summarizes the findings noted among the 170 students examined in this study. Intrathoracic calcification was noted in 41 students, pleural scarring with adhesions in four, mediastinal emphysema in one, and mediastinal emphysema with subcutaneous emphysema in one. Normal anatomical variations noted during the study, such as azygous lobes, accessory lobes, bifid or cervical ribs, and apical (pleural) capping were not tabulated. The incidence of asymptomatic yet detectable extra-alveolar air in these 170 personnel following routine submarine escape training was found to be 1.18 per cent.

TABLE III.
List of Roentgenographic Findings Noted in 170 Personnel Participating in Study.

	FINDINGS	NUMBER NOTED	INCIDENCE
A)	Calcific Lesions (all types)	41	24.1%
	1. Ghon Complex	16	
	2. Ghon Complex with one calcified perihilar lymph node	20	
	3. One calcified perihilar lymph node	4	
	4. Disseminated calcifications compatible with old Histoplasmosis	1	
B)	Pleural Scarring with Adhesions	4	2.36%
C)	Extra-Alveolar Air (all types)	2	1.18%
	1. Mediastinal emphysema	1	
	2. Mediastinal emphysema with subcutaneous emphysema	1	

Of notable significance are the two students found to have asymptomatic mediastinal emphysema. Neither student had any associated intrathoracic calcification, pleural scarring, or pleural adhesions. One of these students did have an associated subcutaneous emphysema which involved a small area of the thorax anterior to the superior portion of the sternum. Both students had PA and left lateral chest roentgenograms and in each instance, the diagnosis of mediastinal emphysema was suggested on the PA view and confirmed on the left lateral view. The 1x15 mm area of subcutaneous emphysema was noted only on the left lateral view and, even in retrospect, could not be visualized on the

PA view. The quantity of mediastinal air noted was judged to be approximately equal for both students. In each case, one radiolucent streak was noted on the PA view at the left portion of the cardiac border, and two such streaks were noted on the left lateral views in the region of the middle mediastinum. Exact measurements of these streaks were unobtainable due to border blending, but none is thought to have exceeded one millimeter in width or twenty millimeters in length.

REVIEW AND SUMMARY

Both a review of the results obtained in this study and a summary of the related studies discussed provide answers to each of the original questions asked by this author.

(1) Does a detectable quantity of extra-alveolar air exist in personnel immediately following uneventful submarine escape training? YES. In this study, two of the 170 personnel examined after such training were found to have roentgenographic evidence of extra-alveolar air. In both patients this air was located in the mediastinum. Subcutaneous emphysema of the anterior thoracic wall was an associated finding in one of these two patients.

(2) Is extra-alveolar air more common after routine submarine escape training than current knowledge would indicate? NO. As previously stated, in the only identical study reported, British physicians found one patient with extra-alveolar air in 70 patients studied, an incidence of 1.43%. This author reported an incidence of 1.18%. The difference between incidences of extra-alveolar air in the two studies was not considered by this author to be statistically significant.

However, this answer must be qualified, since a patient with extra- alveolar air after submarine escape training is considered to represent a casualty due to such training and is recorded as same. In this situation the reported incidence of extra-alveolar air, occurring with or without air embolism, is .008%. Table IV, compiled from Moses' New London studies, shows the incidence of extra-

alveolar air in relation to the three methods of individual submarine escape used in training.[17] Also, for this situation the second question must be answered YES, since a 1.18% incidence of extra-alveolar air after routine submarine escape training is much greater than the .008% incidence which is currently acknowledged as the true incidence of extra-alveolar air after such training.

TABLE IV.

Number and Incidence of Casualties Resulting From Three Methods of Ascent Used in Submarine Escape Training: (for casualties due to extra-alveolar air with air embolism).

ASCENT TYPE	CASUALTIES PER TOTAL ASCENTS	INCIDENCE
S.E.A.	7/193,000	.004%
Free	10/16,500	.061%
Bouyant	1/6,500	.015%
Total (All Types)	18/216,000	.008%

Any comparison of submarine escape training with altitude studies is considered by this author to be invalid if such a comparison relates to the production and incidence of extra-alveolar air. By means of simple conversion of factors, the author calculated that the rate of pressure change is over 350 times greater in submarine escape training than in the altitude studies previously mentioned. A 5.5 foot per second ascent rate for submarine escape training corresponds to a rate of pressure change of -128 mm. of Hg per second.[19] The ascent rate of 1000 feet per minute, as reported by Tomashefski in his altitude studies, corresponds to a rate of pressure change of only -0.36 mm of Hg per second. It can readily be seen that an individual subjected to decreasing pressure due to altitude has a far greater amount of time during which he may release expanding air from his lungs than does an individual undergoing submarine escape training. This author has concluded that the incidence of pneumothorax and other evidence of extra-alveolar air due to pressure changes reported in altitude studies should not be compared with the incidence of extra-alveolar air produced by pressure changes experienced in submarine escape training.

(3) Are pre- and post-ascent chest roentgenograms feasible as a screening procedure for detection of extra-alveolar air resulting from submarine escape training? YES. The findings of this study were quite similar to those of the previously mentioned smaller study conducted by British physicians.[17] It is recommended by this author that both PA and lateral (left or right) chest roentgenograms be obtained for the post-ascent views. This recommendation was based on the following: (1) subcutaneous emphysema may be visible only on a lateral view, (2) most radiologists feel that both a PA and a lateral view are mandatory for the roentgenographic diagnosis of pneumomediastinum,[1,20,21] and, (3) as reported by Cimmino, a paracardiac pneumothorax may mimic mediastinal free air if only a PA view is obtained; this is a rare occurence, however, and usually occurs only if the patient is X-rayed while in the supine position.[1]

Existence of extra-alveolar air was regarded as questionable on 38 of the 170 post-ascent PA films obtained by this author. However, extra-alveolar air was subsequently determined to exist on only two of those 38 films when comparison was made with pre-ascent and/or lateral films. Thus, pre-ascent films are considered to be essential for any future screening procedure which may be attempted.

(4) Is there any correlation between the presence of extra-alveolar air and air emboli in personnel following routine submarine escape training? PROBABLY, YES. As indicated in Moses' overall statistics, air embolism was noted to occur in 16 of 22 patients with extra-alveolar air. Moses' New London statistics reported evidence of air embolism in 13 of 18 patients with extra-alveolar air.[17] This author has postulated that many casualties reported with air embolism also have associated extra-alveolar air, but that this latter entity is either not looked for, not detected, or just not reported. For both entities the pathogeneses are identical. However, as mentioned previously, a critical pressure gradient is required before alveolar air or free air is able to rupture into the circulation and thus produce air emboli. Thus, perhaps the frequent association of air emboli with extra-alveolar air occurs only in obvious or major casualties resulting from submarine escape training and not in the minor or asymptomatic casualties resulting from such training. A definitive answer to this question must await further study.

CONCLUSIONS

1. The incidence of roentgenographically detectable extra-alveolar air in 170 personnel immediately following routine submarine escape training was found to be 1.18%.

2. The production or incidence of extra-alveolar air due to decreasing pressure with increasing altitude cannot be compared to the production or incidence of extra-alveolar air due to submarine escape training due to the much smaller rate of pressure change normally experienced in the former.

3. The incidence of air emboli and/or extra-alveolar air following submarine escape training may be much greater than previously suspected.

4. Extra-alveolar air and air emboli occur together in approximately three of every four reported casualties due to submarine escape training.

5. PA pre-ascent and PA and lateral post-ascent roentgenograms should be used to properly evaluate personnel for extra-alveolar air following submarine escape training.

ACKNOWLEDGEMENTS

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13. ABSTRACT

Submarine escape training for submarine personnel has been conducted by the U.S. Navy since 1930. By 1957, more than 250,000 ascents had been recorded. Most casualties due to this training involved extra-alveolar air and/or embolism, and those few casualties which have occurred had usually been recorded and reported. In view of this, several questions were asked, among them one concerning the incidence of extra-alveolar air after submarine escape training in personnel who are not considered to be casualties.

Chest roentgenograms of 170 personnel taken immediately after undergoing routine submarine escape training were examined. Two men were found to have roentgenographic evidence of extra-alveolar air. The close association of extra-alveolar air with air embolism is postulated and the true incidence of these entities after submarine escape training is questioned. It was concluded that the incidence of air emboli and/or extra-alveolar air following submarine escape training may be much greater than previously suspected.

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